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Paediatrics

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Mithu.

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GROWTH & DEVELOPMENT

Growth is an increase in body size. It is a continuous process starting in the intrauterine life and continuing throughout childhood. Development refers to acquisition of various skills appropriate ages.

Growth

Periods of growth:

1. Intrauterine:
    a. ovum- 0 to 14 days
    b. embryo – 14 days to nine weeks
    c. fetus – nine weeks to birth
    d. perinatal – 22 weeks to seven days
2. Extrauterine :
    a. Neonatal: 0 to 28 days
    b. in fancy – 29 days to one year
    c. toddler – 1 to 3 years
    d. preschool – 3 to 6 years
    e. school – 6 to 12 years
    f. adolescence – 10 to 19 years
        (a) early – 10 to 13 years
        (b) mid – 14 to 16 years
        (c) late – 17 to 19 years

Factors affecting growth

1. genetic factors
2. nutrition
3. chronic diseases
4. Hormones: important hormones for intrauterine growth are thyroxine (secretion starts at 12 weeks of intrauterine life), insulin and glucocorticoids. After birth growth hormone is the most important hormone throughout childhood. Thyroxine is also important. Gonadal hormones are important for pubertal spurt.

Assessment of growth

Done by monitoring of anthropometric parameters mainly weight, length, head circumference.

Age	**Birth**	**Three months**	**Five months**	**One year**	**2 year**	**Three years**	**Five years**
Weight	3 kg		Double – 6 kg	Triple – 9 kg	4X 12 kg	5X 15 kg	6X 18 kg
Length	50 cm	60 cm	65 cm	75 cm	84 cm		100 cm at four years
Head circumference	35 cm	40 cm		45 cm	48 cm		52 cm at 12 years

Rate of gain

age	0 to 3 months	3 to 6 months	6 to 9 months	9 to 12 mnth	Subsequently
Weight	30 g per day	20 g per day	15 g per day	12 g /day	6 to 8 g per day
Length	3 cm/ month	2 cm/month	1.5 cm/ mnth	1.2 cm/month	1 cm/month
Head circumference	2 cm/ month	1 cm/month	.5 cm/month	.5 cm/month	

Growth charts

They help to assess growth and diagnose deviations. Normal range is conventionally taken between third and 97th percentile or mean ± 2 standard deviations.

NCHS charts (1977): National centre for health statistics-based on formula fed US population.

CDC charts (2000). Centre for disease control and prevention

WHO MGRS charts-published in 2006 and presently recommended for use

1. Multicenter growth reference study (MGRS).
2. Representative of developing countries.
3. Sample from Brazil, Ghana, India, Norway, Oman & United States.
4. Breastfed, good socio economic class.
5. Extra indicators: skin fold thickness and BMI

Different tissues of body grow at different rates

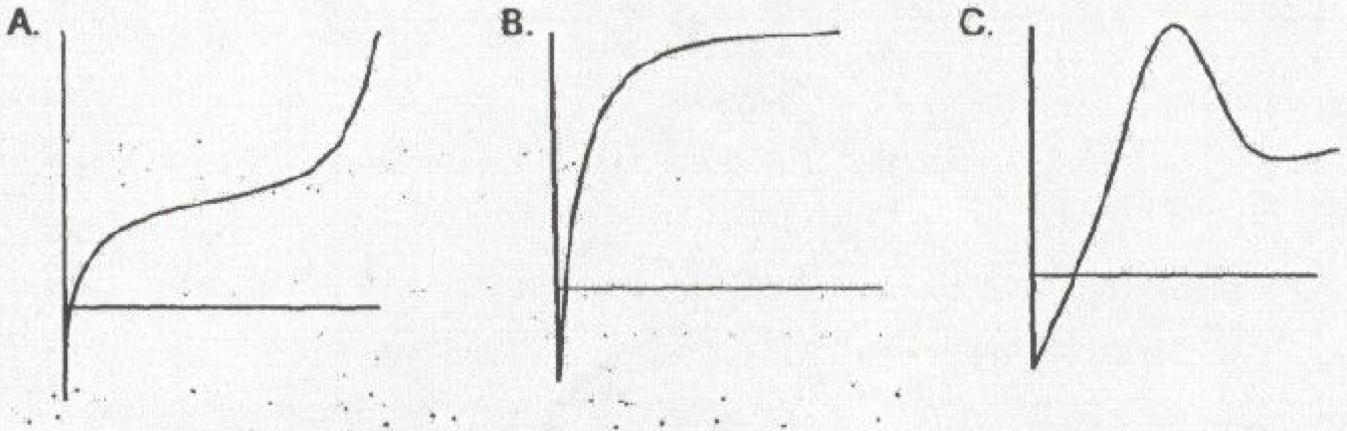
A. General growth-initial spurt in the first two years—→slowing-→pubertal spurt
B. brain growth – maximum in first two years
C. lymphoid growth – maximum between 4 to 8 years
D. gonadal growth – during puberty
E. dental growth –
    a. primary dentition

Central Incisors	Lateral Incisors	First Molar	Canine	Second Molar
6 months	one year	15 months	18 months	20 to 30 months

Secondary dentition

Mnemonic: *mummy is in pain papa can make medicine*

First Molar: Central Incisor: Lateral Incisor: First Premolars: Second Premolars: Canine: Second Molar: Third Molar//6:7:8:9:10: 11: 12: 18 (age in years)



Short stature

Height <3rd centile/ 2 standard deviations

Based on Upper segment/lower segment ratio, classified as proportionate and disproportionate

Normal Upper segment to lower segment ratio:

1.7 at birth 1.3 at three years at six years, at 10 years & 0.9 in adults

Causes:

Proportionate short stature (Normal US/LS ratio)

Normal variants

1. Familial
2. Constitutional

Pathological

1. Symmetrical intrauterine growth Restriction
2. Torch infections
3. Chromosomal abnormalities like Down's syndrome, Turner's syndrome, Prader-Willi syndrome, Noonan syndrome, Russell Silver syndrome, Seckel syndrome
4. Malnutrition
5. chronic diseases
6. endocrine
7. psychosocial

Disproportionate short stature

Increased US/LS, ratio (short limbs)

1. Rickets
2. Achondroplasia
3. Cretinism(untreated, congenital hypothyroidism)

Decreased US/LS ratio (short trunk)

1. Vertebral anomalies
2. Mucopolysaccharidosis
3. Spondyloepiphyseal dysplasia

Mid parental height or predicted adult height also helps to find the cause

Bone age should be calculated and may be helpful in finding aetiology

Most useful x-ray for bone age between 1 to 13 years is hands and wrists. Calculation is done with the help of Tanner Whitehouse scoring. Between 3 to 9 months shoulder joint x-rays preferred and between 12 to 14 years elbow and hip x-rays may be useful.

Bone age is generally the same as height age and less than chronological age in most cases. It is markedly delayed in hypothyroidism and growth hormone deficiency. In familial short stature, it is same as chronological age. In precocious puberty, bone age exceeds chronological age.

Most common cause of short stature in children is *constitutional growth delay*. These children are normal at birth, grow normally for 6 to 12 months and then their growth slows. The height velocity again accelerates around 2 to 3 years and they grow at a normal velocity. Onset of puberty is the late but final height is normal. *Familial short stature:* the child is short because the parents are short.

Adolescence:

Early adolescence: 10 to 13years
Mid adolescence: 14 to 16 years
Late adolescence: 17 to 19 years

Primary sexual characters: testicular and penile growth in boys and breast and uterine growth in girls. These changes are produced by the sex hormones estrogens in girls and testosterone in boys.

Secondary sexual characters: pubic and axillary hair growth, acne, axillary odour. These changes are produced by adrenal androgens.

Pulsatile GnRH secretion-----> increased gonadotropins----> increased sex hormones

Staging of puberty is done by Tanner's staging system and divided into stage I to V. this is also called the *sexual maturity rating* (SMR stages I to V)

Puberty in females:

Starts around the age of 10 years and is completed in five years.

Thelarche (breast development) ----> Pubarche (pubic hair development) --->Menarche (onset of menstrual cycles). The time gap between breast development and menarche is usually 2 to 3 years. Growth spurt is early and occurs just before menarche (SMR stage II-III).

Puberty in males:

Starts around 11 to 12 years and is complete by 14 to 15 years

Testicular enlargement --→penile enlargement and pubic hair development----→spermarche

Growth spurt is late (SMR stage III-IV).

Development:

GROSS MOTOR:

	Ventral suspension	Prone	Pull to sit	Standing
4 weeks		Momentarily lifts chin off couch; Head predominantly to one side.	Almost complete head lag, back rounded	

6 weeks	Head held up momentarily in same plane as rest of body	Chin raised intermittently off couch. Head turned to one side	Head lag considerable	
12weeks	Head held up beyond plane of rest of body	Plane of face at angle of 45 to 95 degrees from couch, weight borne on forearms with shoulder off the couch	Slight head lag	
20weeks	-		Complete neck control; plays with feet	
5months	-	Rolls prone to supine	Sit with external support	
6months	-	Rolls supine to prone	Sit with support	Bears weight when made to stand
8moths	-	Crawls	Sit without support	
9months			Pivoting	Stand with support and pull to stand
10months	-	Creeps		Cruising around furniture
1 year				Stand independently
13-15 months				Walks independently
18months				Crawls up and down stairs, pull a toy; runs
2years				Goes up and down stairs alone, 2 feet per step. Walks backward in imitation, kicks ball without overbalancing; jumps;
3years				goes up stairs, one foot per step and down stairs two feet per step, rides tricycle
4years				Goes down stairs, one foot per step. Hops on one foot

Fine Motor, language, social and vision

	Fine motor	Language	Social	Vision
6 weeks			Social smile	Follows moving person through 90⁰
10-12 weeks		Cooing/vocalisation		
12 weeks	Reflex grasp goes	Turns head to sound	Recognizes mother	Hand regard; follows object through 180⁰
4 months	Reaches for objects but overshoots; bidextrous reach; holds objects given in hand	Laughs aloud		
5 months		Ah-goo sounds		Hand regard goes
6 months	Unidextrous reach; transfers objects; ulnar grasp; mouthing	Mono syllables speech	Smiles at mirror image; stranger anxiety; responds to no;imitates simple gestures	
9 months	Immature pincer grasp	Bi syllables	Bye-bye	Responds to name
1 year	Mature pincer grasp; Mouthing goes; drinks from cup with spilling	1-2 words with meaning		
15 months	Imitates scribbling; tower of 2 cubes; drinks without spilling; turns 2-3 pages	Jargon		
18 months	Eat with spoon; tower of 3 cubes;	8-10 words	Follows simple orders; domestic mimicry	
2 years	Tower of 6 cubes; train of blocks; vertical and circular stroke;	2-3 word sentences; uses pronouns; vocabulary 100	ready for toilet training; indicates toilet needs	

	turn pages one at a time; undress; can wear shoes or socks	words		
3 years	Copies circle; bridge with blocks; tower of 9 blocks; dress and undress fully except buttons	Asks questions	Knows name and sex; identifies 1-2 colours; simple rhymes	
4 years	Copies cross, plus and rectangle; gate with blocks	Tells stories	Right & left discrimination; imaginative play	
5 years	Copies triangle; ties shoelaces		Identify 4 colours; repeat 4 digits	

Pica

Defined as eating of non nutritive substances for at least one month. Common in children less than five years of age. Co existent iron deficiency anemia and parasitic infestations may be there.

Enuresis

Normal age of toilet training is around two years and most children are toilet trained by five years. Urinary incontinence beyond the age of four years for daytime and six years for night-time or loss of continence after at least three months of dryness. Most common is primary nocturnal incontinence. This is usually idiopathic; a strong family history may be present.

Treatment includes behavioral therapy, alarms, and drugs (desmopressin, imipramine, Oxybutynin)

Breath holding spells

Seen in children between six months to 5 years of age. Provoked by excessive crying after which the child holds breath, stops breathing and becomes cyanosed or pale. Some may have seizure. Treatment is to give a strong physical stimulus at the onset of spell. Iron supplementation in anemic children should be done.

Attention deficit hyperactivity disorder

Most common neurobehavioral disorder of school age children. More common in boys. Clinical features include inattention, impulsivity, hyperactivity, emotional lability and difficulty in organizing tasks. The behaviour must begin before seven years of age and should be present for at least six months in two different settings. Treatment includes behavioural therapy and drugs like methylphenidate, amphetamines.

Dyslexia

It is the most common learning disability. There is Difficulty in accurate word recognition and decoding. Behaviour therapy and special education are required.

Stuttering is common and physiological between 2 to 5 years of age and reassurance should be given.

Autism

It is one of the pervasive developmental disorders, the other two being asperger syndrome and rett syndrome. Diagnosis can be done by 18 months of age. It is characterised by an impairment in verbal and non-verbal communication and in social interactions. These children have a poor eye contact, delayed speech, stereotypical body movements and solitary play. Seizures may be present. IQ may be marginally less.

Reflexes

REFLEX	ONSET	FULLY DEVELOPED	DURATION
Palmar grasp	28 wk	32 wk	2–3 mo
Rooting	32 wk	36 wk	Less prominent after 1 mo
Moro	28–32 wk	37 wk	5–6 mo
Tonic neck	35 wk	1 mo	6–7 mo
Parachute	7–8 mo	10–11 mo	Remains throughout life
Landau	6-8 mo	-	2 years

NEONATOLOGY

Neonatal period: birth to <28 days (early neonatal period- birth to <7 d; late neonatal period- 7-27 days)

Post neonatal period: 28 days to less than one year

Perinatal period: 22 weeks to less than seven days

Term newborn: 37 wks- <42 wks

Preterm: <37 wks

Postterm: >=42 wks

Low birth weight: <2500g

Very low birth weight: < 1500g

Extremely low birth weight: <1000 g

CARE AT BIRTH AND RESUSCITATION

- Normal newborn establishes good crying effort spontaneously.
- *Five cleans:* Clean hands, Clean delivery surface, Clean cord cut, Clean cord tie, Clean cord stump.
- Temperature maintenance very imp.(discussed later)
- *Rule out major anomalies*- TEF (pass a feeding tube into stomach); check cord for 2 arteries and 1 vein (single UA assos with GI and renal anomalies)
- Inj vitamin K intramuscular 1 mg
- Initiation of breastfeeding as soon as possible.

Resuscitation:

Resuscitation is done in accordance with neonatal resuscitation programme (NRP). The sequence of resuscitation in newborns is TABC with temperature taking precedence over airway, breathing and circulation. Temperature maintenance is of utmost importance as a hypothermic baby is difficult to resuscitate.

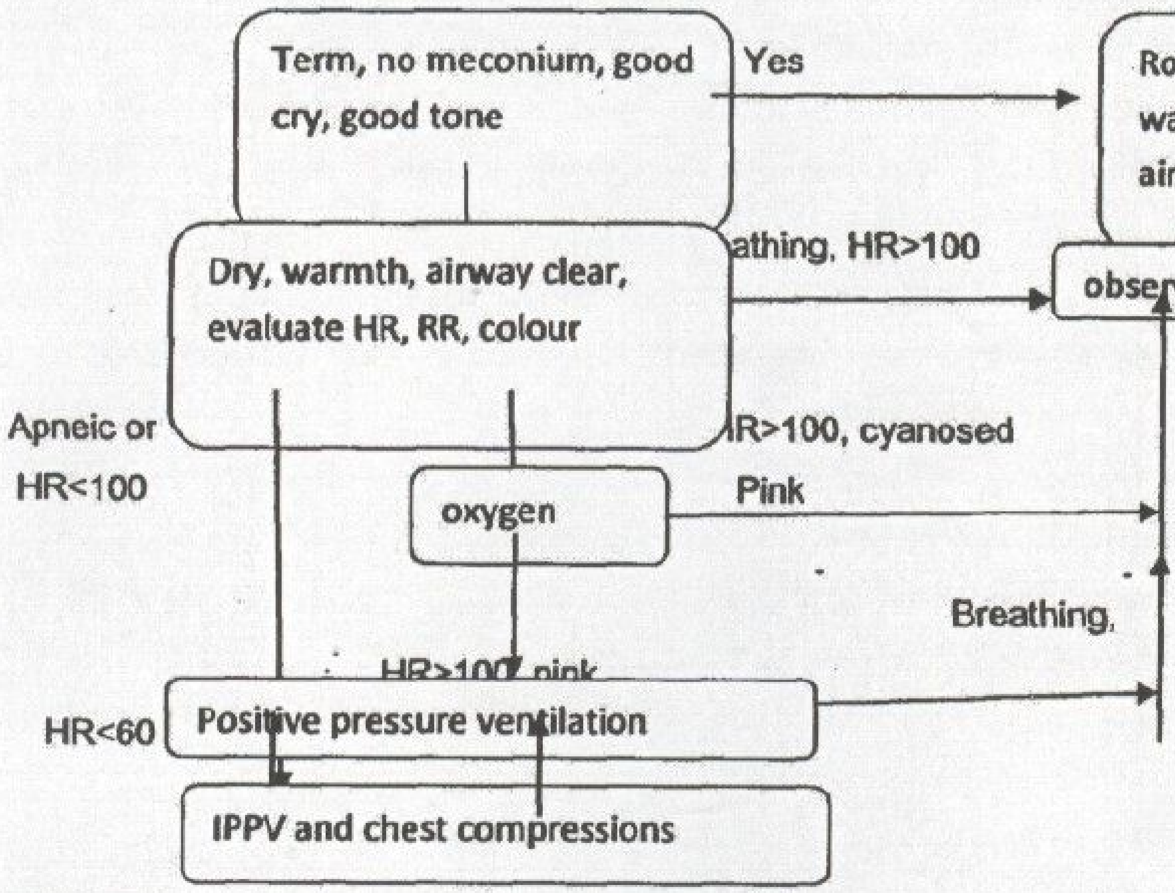
Most babies will establish a good spontaneous cry and respiration after cord clamping. Some babies may be apneic or have gasping respiration. Such babies will require assistance in establishing hemodynamic stability.

Resuscitation equipment includes self inflating ambu bag with mask (capacity 250-750 ml), laryngoscope with straight blades, endotracheal tubes, oxygen source, reservoir, drugs (adrenaline, naloxone, and normal saline), and suction equipment.

Endotracheal tube sizes: <1000 g-2.5 Fr
1000 to 2000 g- 3.0 Fr
2000 to 3000 g - 3.5 Fr
> 3000 g- 4.0 Fr

100% oxygen used for resuscitation.

RESUSCITATION ALGORITHM


Term, no meconium, good cry, good tone
Yes
Routine care- warmth, clear airway, dry
athing, HR>100
Dry, warmth, airway clear, evaluate HR, RR, colour
observe
Apneic or HR<100
R>100, cyanosed
Pink
oxygen
Breathing,
HR>100 pink
HR<60
Positive pressure ventilation
IPPV and chest compressions

** Assessments and decisions made every 30 seconds

Routine prolonged suction not recommended and is better avoided as it can cause bradycardia and apnoea.

Babies with meconium stained liquor if non-vigorous require direct endotracheal suctioning and should be intubated at birth.

Techniques for chest compression- thumb technique (better) and two finger technique.

Apgar score not needed to determine the need for resuscitation or the steps necessary for resuscitation. It indicates how well the baby is responding to resuscitation.

APGAR SCORE:

SIGN	0	1	2
HR	Absent	<100	>=100
Respiration	Absent	Weak, irregular	Good cry
Tone	Limp	Some flexion	Active
Reflex	None	Grimace	Cough, gag
Colour	Central cyanosis	acrocyanosis	pink

NORMAL NEWBORN:

Vital signs: heart rate: 110-160, respiratory rate- 40-60 breaths per minute, temperature- 36.5-37.5. Temperature should be measured in the axilla for three minutes. Respiratory distress is indicated by respiratory rate> 60, grunting, and retractions.

Anthropometry: length- 50 cm, head circumference 33 to 37 cm, chest circumference 3 cm less than head circumference (in IUGR babies this difference is> 3 cm)

The following features/phenomena are normal in a newborn

Acrocyanosis: peripheral cyanosis of hands and feet is normal in first few hours in newborns.

Caput succedaneum: scalp swelling due to subcutaneous edema on the presenting part. It is caused by the venous engorgement during labor. This should be differentiated from a cephalhematoma which is due to trauma and bleed in the subperiosteal plane.

Erythema toxicum: it is a rash seen in normal newborns. Appears on the second to third day starting on the face and trunk. Usually resolves spontaneously in a week to 10 days. Eosinophilic infiltrate.

Epstein pearls: retentions cysts usually seen on palate

Subconjunctival hemorrhages, stork bites(capillary hemangioma on eyes and forehead)

Withdrawal bleeding: female newborns may have vaginal discharge or vaginal bleeding in the first week due to withdrawal of maternal hormones.

Physiological weight loss: newborns may lose up to 7-10% of birth weight in the first few days. They regain the birth wt. by around tenth day and thereafter start gaining weight.

Physiological jaundice: may be seen in the 60% of normal newborns. Starts on second or third day, peaks on the third to fifth day, generally resolves by 7 to 10 days.

Transitional stools: increased frequency with greenish loose stools seen between third to fifth day of life.

Breast discharge or enlargement may be seen which spontaneously resolves.

The cord should be kept dry, falls in 7 to 10 days.

BREASTFEEDING

Normal newborns should receive exclusive breastfeeding for first six months of life.

Advantages of exclusive breastfeeding

- initial milk called colostrum is rich in antibodies especially IgA which provides local respiratory and gastrointestinal mucosal immunity
- less allergies
- growth is better
- intelligence quotient of exclusively breastfed babies found higher
- lesser incidence of infections in childhood
- Benefits to mother: helps in uterine involution, less PPH, contraception, decreases chances of breast and ovarian cancer, and helps in weight shedding.

Milk production mainly by prolactin and milk ejection by oxytocin. Main stimulus for both is sucking by the baby.

Colostrum is the initial milk in first 3-4 days and is rich in antibodies and vitamins A, D, E, K. Transitional milk is produced after colostrum and mature milk starts in around two weeks.

Mothers with preterm babies have milk which is richer in proteins, sodium, iron, antibodies and calories (preterm milk)

Foremilk is the initial part of milk which satisfies the baby's thirst. It is more watery and has more proteins. *Hind milk* is richer in fat and calories and satisfies the baby's hunger.

Breast milk provides 7 g carbohydrate and 1 g protein per 100 ml. The breast milk sugar is lactose.

Birth asphyxia

Defined by WHO on basis of APGAR at one minute. A score of 4 to 7 is moderate birth asphyxia and a score of 0 to 3 is severe birth asphyxia.

American Academy of Pediatrics defines severe birth asphyxia as cord blood pH less than 7, APGAR score 0 to 3 at more than five minutes or features of end organ dysfunction present in the baby.

Manifestations of birth asphyxia: almost all organ systems suffer damage. Renal damage is the most common manifested as oliguria, edema and in severe cases acute renal shutdown. Hypoxic ischaemic encephalopathy may occur. There may be metabolic complications like hypoglycemia, hypocalcaemia, hyponatremia and metabolic acidosis. Respiratory system: meconium aspiration syndrome, persistent pulmonary hypertension; necrotizing enterocolitis; thrombocytopenia or disseminated intravascular coagulation.

Neonatal seizures:

- manifested as focal clonic or subtle seizures
- common causes: birth asphyxia, hypoglycemia, hypocalcaemia
- best prognosis: hypocalcaemia seizures
- anticonvulsant of choice: phenobarbitone

Neonatal sepsis:

- maybe early onset (less than 72 hours) or late onset (after 72 hours)
- early onset generally seen when mother has history suggestive of chorioamnionitis
- late onset generally originates from nursery or community
- common causes are gram negative organisms like E.Coli, Klebsiella, Group B streptococcus, Listeria, Staphylococcus aureus
- clinical features include lethargy, refusal to feed, sclerema, hemodynamic instability, apneas
- *Sepsis screen:* includes total leukocyte count(<5000), absolute neutrophil count(<1800-the best indicator), immature to total neutrophil ratio(> 0.2), C-reactive protein (>1 mg%) and microESR (age in days +3). If two or more of these are positive, a provisional diagnosis of sepsis is made

Low birth weight

Babies less than 2500 g at birth. May be preterm or IUGR or both.

IUGR: weight less than 10th percentile for gestation. chest circumference more than 3 cm less than head circumference. May be symmetrical or asymmetrical;

Symmetrical IUGR: causes-genetic, chromosomal anomalies, TORCH infections; ponderal index(weight g/length cm cube *100)>=2

Asymmetrical IUGR: causes-maternal diseases, PIH, placental insufficiency; ponderal index<2

Features of maturity

- skin: gelatinous pink translucent in preterm with visible vessels. Lanugo is abundant. IUGR babies have more mature skin with sparse lanugo.
- breast bud: poorly developed with faint areola in preterm and well-developed in IUGR.
- Ear cartilage : poorly developed with poor recoil in preterm. well-developed with a good recoil in IUGR.
- Sole creases: up to anterior one third in preterm and more in IUGR.

- Genitalia: female: poorly developed majora with visible minora and clitoris in preterm, in IUGR majora covers minora and clitoris well. Male: poorly developed scrotum with undescended testes in preterm, well-developed scrotum with good rugosities and descended testes.
- Expanded new ballards score is a scoring method to determine maturity.

Complications of low birth weight: birth asphyxia, hypothermia, infections, hypoglycaemia, hypocalcaemia. In preterms, respiratory distress syndrome, intraventricular haemorrhage, necrotising enterocolitis, retinopathy of prematurity, apnoeas of prematurity.

Feeding of LBW:

>34 weeks, >1800 g- breastfeeding;

1200-1800g, 30-34 weeks- spoon or gavage;

<1200, <30 weeks- i.v.fluids

Hypothermia and temperature maintenance:

Newborns are susceptible to hypothermia because of higher body surface area to weight ratio. Heat loss at birth occurs by conduction, convection, radiation and evaporation.

Normal newborns maintain temperature with the help of Brown fat(non shivering thermogenesis). This adipose tissue present in *axilla, around adrenals, perirenal area and periscapular area*. Brown fat is deficient in preterm babies and IUGR babies making them more prone to hypothermia.

Normal temperature- 36.5 to 37.5°C

1. Cold stress (mild hypothermia)- 36 to 36.5°C; hands and feet cold abdomen warm
2. moderate hypothermia- 32 to 36°C; both peripheries and abdomen cold
3. severe hypothermia - <32°C

Warm chain: set of 10 steps in the delivery room to maintain the temperature of the newborn.

Kangaroo Mother Care (KMC): continuous skin to skin contact between mother and healthy baby to maintain temperature. Used for care of low birth weight babies.

Treatment of hypothermia: rapidly warming till 34°C and then slowly warming.

Mechanism of temperature maintenance: incubator- convection; radiant warmer-radiation.

Temperature range in which the basal metabolic rate of babies is minimum is called thermoneutral zone and depends on the age and weight of the baby.

Respiratory distress syndrome (hyaline membrane disease)

- seen in preterm babies less than 34 weeks(10-15% incidence) though older babies may have this especially with birth asphyxia, diabetic mother, caesarean delivery, acidosis.
- Surfactant deficiency (normally production starts at 20 weeks, peaks at 35 weeks)
- most common cause of respiratory distress in preterm babies within the first six hours
- x-ray shows reticulogranular pattern, ground glass appearance, low-volume lungs, air bronchogram or whiteout lungs
- shake test on amniotic fluid may give an idea about lung maturity

Transient tachypnea of newborn (TTN)

- is due to delayed clearance of lung fluid
- mostly seen in term babies delivered by Caesarean section
- generally a mild respiratory distress that settles in 6 to 12 hours

- x-ray may show hyper expansion with prominent vascular markings, fluid in interlobar fissure

Hypoglycaemia

- blood glucose less than 40 milligram/decilitre
- Common in low birth weight, infant of diabetic mother, large for gestational age, Rh isoimmunisation, sick babies

Hypocalcaemia

- total Calcium < 7 mg/dl or ionised calcium < 4
- early onset hypocalcaemia (less than 72 hours): in preterms less than 32 weeks, infants of diabetic mother, birth asphyxia, maternal hyperparathyroidism
- late onset (more than 72 hours): mothers with vitamin D deficiency, babies on anticonvulsants, cow's milk fed

Apnoea

- cessation of breathing for >= 20 seconds or less than 20 seconds but with bradycardia or cyanosis
- apnoea of prematurity seen in preterm babies between second to fifth day, treatment is aminophylline, caffeine.

Infants of diabetic mothers

- Macrosomia
- more chances of Hyaline membrane disease
- hypoglycaemia, hypocalcaemia, hyper bilirubin anaemia
- polycythemia
- congenital anomalies especially cardiovascular
- cardiomyopathy and persistent pulmonary hypertension
- lazy left colon syndrome

Necrotising enterocolitis:

Seen in preterm babies after first week of life, almost always in formula fed. Clinical features similar to septicaemia with abdominal distension and feed intolerance. X-ray will show bowel distension and pneumatosis intestinalis

Hemorrhagic disease of newborn

- due to vitamin K deficiency
- early onset (<3d): due to maternal drugs like anticonvulsants, INH, anticoagulants
- classical(3-7d): almost always in breastfed babies due to inadequate vitamin K
- late onset(>2wk): due to hepatic disorders or malabsorption syndromes
- early onset and classical prevented by vitamin K given at birth
- early onset prevented by vitamin K given to mother

Neonatal cholestasis

- direct bilirubin more than 2 mg per decilitre or more than 20% of total bilirubin
- main causes are extrahepatic biliary atresia (EHBA) & neonatal hepatitis.
- there is jaundice, highly coloured urine and pale stools
- EHBA to be corrected as soon as possible to prevent irreversible damage
- EHBA: HIDA scan shows non-excretion of contrast, high GGT levels, suggestive liver biopsy

(ductular proliferation and fibrosis).

- liver biopsy in neonatal hepatitis: necrosis, ballooning and giant cells
- peroperative cholangiography is the gold standard

TORCH Infections or congenital Infections

- transmitted from mother to baby
- clinical features include growth retardation, microcephaly on hydrocephalus, hepatosplenomegaly, anaemia, thrombocytopenia, hearing loss, ocular problems

TOXOPLASMOSIS

caused by Toxoplasma gondii

transmission rate increases with gestation

classical triad: chorioretinitis, Hydrocephalus, cerebral calcification

treatment for baby: pyrimethamine-sulfadoxine and treatment for mother: spiramycin

RUBELLA

transmission maximum in first trimester and decreases with gestation

congenital rubella syndrome: cataract, sensorineural hearing loss, congenital heart disease(PDA, VSD, Pumonary stenosis)

hearing loss is the most common

eye defects: retinopathy (salt and pepper retinopathy) more common than cataract

CYTOMEGALOVIRUS

most common congenital infection

transmission at all stages of gestation as well as postnatally

cytomegalic inclusion disease: microcephaly, chorioretinitis,hepatosplenomegaly, jaundice, petechie.

treatment is ganciclovir

HERPES SIMPLEX

transmission is perinatal

mostly caused by HSV 2

most common manifestation is encephalitis and viral septicaemia in the newborn

treatment is intravenous acyclovir

CONGENITAL SYPHILIS -

- transmission occurs when mother has primarily on secondary syphilis at any gestation
- fetal or neonatal death can occur
- early signs: (first 2 years) haemolytic anaemia, thrombocytopenia, hepatosplenomegaly, jaundice, lymphadenopathy, OSTEOCHONDRITIS AND PERIOSTITIS, mucocutaneous rash, snuffles
- late signs (first 2 decades): bony deformities, Hutchinson teeth, mulberry molars, saddle nose,rhagades, clutton joints
- diagnosis is by serological tests
- treatment is penicillin

RESPIRATORY SYSTEM

Common cold: causative organisms-adenovirus, influenza virus, rhinovirus, parainfluenza virus and RSV (Respiratory Syncytial virus)

Acute tonsillopharyngitis : may be viral. Important bacterial cause is Group A beta haemolytic streptococci

Herpangina: caused by Coxsackie A. Pharangitis with papulovesicular mucosal lesions

Pharyngoconjunctival fever: caused by adenovirus

Acute epiglottitis:

- Usually bacterial, mostly caused by **H.influenzae** .
- High fever, croupy cough, dysphagia respiratory distress, stridor.
- Diagnosis is by laryngoscopy(inflamed epiglottis visible) or by X-Ray
- Treatment—Hospitalisation, humidified oxygen, **antibiotics** like ampicillin

Acute laryngotracheobronchitis or Viral croup

- Viral: Most common- Parainfluenza type 1; others- adenovirus, influenza virus, rhinovirus, parainfluenza virus type 2 and 3 and RSV (Respiratory Syncytial virus)
- Child has croupy or brassy cough, mild fever, stridor and in severe cases respiratory distress
- X Ray may help in diagnoses
- Symptomatic treatment given. Severe cases may need hospitalisation. Single dose of steroids, nebulised epinephrine or budesonide may help.

Pneumonia

Causes :

1) Viral— RSV, adenovirus, influenza virus,parainfluenza virus
2) Bacterial— < 2 months—E.coli, klebsiella, staphylococci
   3 mnths to 3 years—H. Influenza, pneumococcus and staphylococcus
   >3 yr-- pneumococcus and staphylococcus
   Atypical— Chlamydia and Mycoplasma

Clinical features: fever, cough, respiratory distress, vomiting, abdominal pain

- Pneumococcus causes lobar pneumonia
- Staphylococcus causes severe pneumonia with suppuration and bronchoalveolar destruction. Pnematoceles characteristic on X Ray. Pneumothorax common. Most common cause of empyema in chidren <2 years
- Treatment—appropriate antibiotics. Atypical pneumonia responds to macrolides or tetracyclines.

ARI control programme

Acute lower respiratory infections are a leading cause of mortality in children under five years. WHO has recommended clinical criteria for diagnosis of pneumonia in children at primary healthcare level for control of pneumonia deaths.

Signs and symptoms	classification	therapy
Cough or cold no fast breathing no chest indrawing no indicators of severe illness	No pneumonia	Home remedies
Fast breathing present less than two months:> 60/min; 2 to 12 months> 50/min; 12 to 60 months> 40/min	Pneumonia	Antibiotics (cotrimoxazole)
Chest indrawing	Severe pneumonia	IV antibiotic and refer
Cyanosis, severe chest indrawing, inability to feed	Very severe pneumonia	IV antibiotic and refer

Bronchiolitis:

- Age gp- 1 to 6 months, possible upto 2 years
- Cause- mostly RSV. Others-, adenovirus, influenza virus,parainfluenza virus
- Fever, cough, coryza followed by respiratory distress which can be severe and can cause death.
- X Ray –hyperinflation and infiltrates
- Symptomatic treatment. Warm humidifeied oxygen, inhaled bronchodilators may help. Nebulised Ribavirin in those with immunodeficiency, congenital heart disease or chronic lung disease

WHEEZING IN CHILDREN

Causes:

- Asthma
- Wheezing associated lower respiratory illness (WALRI)
- Foreign bodies
- Tropical eosinophilia

ASTHMA

- increased responsiveness of the airways to various stimuli
- widespread narrowing of the airways causing paroxysmal dyspnoea wheezing or cough
- obstruction to the air flow is reversible either spontaneously or after treatment

Symptoms include recurrent attacks of wheezing, cough and breathlessness in severe cases. Symptoms may occur intermittently when it is called mild intermittent asthma or maybe persistent with intermittent acute exacerbations. Acute attacks may be of mild, moderate or severe intensity. Severe attacks are characterised by cyanosis, silent chest and pulses paradoxus.

Investigations:

Diagnosis is mostly clinical. Pulmonary function tests may play an important role in doubtful cases and in monitoring of response to treatment.

The important parameters in spirometry include PEFR and FEV1/FVC ratio (in asthma ratio is less than 0.8).

Treatment:

Intermittent asthma is treated with bronchodilators most commonly beta-2 agonists

Persistent asthma is treated with inhaled corticosteroids. Commonly used steroids include beclomethasone, budesonide and fluticasone.

The drugs are preferably given by inhaled route and a metered dose inhaler (MDI) is used. Children less than 12 years of age may require a spacer with MDI. Children below 4 years of age usually need a spacer with mask with MDI.

Treatment of acute exacerbation: asthma is marked by acute exacerbations which can be of varying severity-mild, moderate or severe.

Feature	mild	Moderate	Severe
Colour	Normal	Normal	Pale or blue
Sensorium	Normal	Anxious	Agitated
Respiratory rate	Increased	Increased	Increased
Dyspnoea	Absent, can speak sentences	Moderate, can speak in phrases	Severe, difficulty in speech
Rhonchi	expiratory	expiratory plus inspiratory	Maybe absent

Treatment

Mild attacks: inhaled beta2 agonists by nebuliser or MDI with spacer.

Moderate to severe attacks: inhaled beta2 agonists frequently plus oral corticosteroids.

Status asthmaticus: oxygen inhalation, subcutaneous terbutaline or epinephrine injection, inhaled salbutamol, injectable steroids as soon as possible. If no improvement, theophylline infusion. Magnesium sulphate infusion can also be used. Mechanical ventilation is required in nonresponsive cases.

Foreign body aspiration

- age-group 1 to 4 years
- history of choking episode may be present thereafter symptoms depend on the nature of foreign body and the site of obstruction.
- The most common symptom is cough
- laryngeal foreign body will manifest as cough, hoarseness of voice and stridor
- foreign bodies present in bronchi may lead to partial or complete obstruction. Symptoms include cough, recurrent wheezing and pneumonia. Collapse can occur.
- X-ray may show hyperinflation or collapse
- treatment is removal of foreign body with a rigid bronchoscope.

CARDIO VASCULAR SYSTEM

Development of heart starts around third week of intrauterine life and a four chambered heart is formed by six-weeks.

Fetal circulation:

Umbilical vein ----> portal vein-----> ductus venosus----> inferior vena cava-----> right atrium-----> left Atrium+ right ventricle----->aorta through left ventricle or ductus arteriosus

Changes at birth:when the cord is clamped, the pressures in the left-sided circulation rise leading to closure of atrial septal defect. Increase in oxygenation is the stimulus for closure of ductus arteriosus. Functional closure of the ductus occurs in around 48 hours and anatomical closure takes around 10 days. It forms the ligamentum arteriosum. The ductus venosus closes and forms ligamentum teres.

Congenital heart diseases

Nadas criteria:

To evaluate for presence of heart disease

Major criteria:

- systolic murmur(>=gr III),
- diastolic murmer,
- cyanosis,
- congestive heart failure

Minor criteria:

- systolic murmur(< gr III),
- abnormal second sound,
- abnormal ECG,
- abnormal x-ray,
- abnormal blood pressure

presence of one major or two minor criteria indicate heart disease

Classification:

Acyanotic heart disease or left to right shunt

ASD, VSD, PDA

- these patients have CHF around 4 to 6 weeks of age
- they have frequent chest infections
- no cyanosis
- x-ray shows plethoric lung fields

Cyanotic heart disease

a. *decreased pulmonary blood flow (right to left shunts):* TOF, tricuspid atresia, Ebsteins anomaly, Fallot's physiology: these patients have cyanosis, clubbing, polycythaemia, cyanotic spells

b. *normal or increased pulmonary blood flow(mixing lesions):* TGA, TAPVC, Truncus arteriosus: these have cyanosis as well as CHF

Obstructive lesions

Coarctation of aorta, Aortic Stenosis, Pulmonary Stenosis

Atrial Septal Defect

3 types:

- Ostium secundum(more common): midportion of atrial septum/at fossa ovalis
- Ostium primum: defect in low atrial septum (endocardial cushion defect)
- Sinus venosus

assoc syndrome: holt oram syndrome, Downs syndrome

Clinical Manifestations

- **Usually asymptomatic**
- RV heave often present
- Systolic ejection murmur in Pulmonic area, no shunt murmer
- **S2 wide and constantly split**
- Eisenmenger physiology not before 20 yrs

Treatment

- Treat CHF
- Infective endocarditis rare, **prophylaxis not needed**
- May spontaneously close.
- Surgical closure indicated in all symptomatic patients and asymptomatic patients with Qp/Qs ratio> 2. Small asymptomatic ASD not closed

Ventricular Septal Defects

Most common congenital defect

Membranous or muscular, **membranous more common**

Clinical Manifestations

- symptoms start around 6 to 10 weeks of age
- Larger, gives growth failure, CHF, recurrent lower respiratory infection
- **pan systolic murmur** at lower left sternal border (pansystolic murmer only in VSD,MR,TR). Smaller defect = louder murmur, asymptomatic
- S2 may be widely split but variable
- x-ray shows cardiomegaly with pulmonary plethora
- Patients with large defects develop pulmonary hypertension and Right ventricular hypertrophy with the reversal of shunt (Eisenmenger syndrome)

Treatment

- Most close without intervention
- Rx CHF
- IE common, prophylaxis is required
- May require surgery -**failure of medical treatment, pulmonary hypertension, Qp/Qs ratio> 2**

Patent Ductus Arteriosus

Connects Pulmonary artery to Aorta just distal to Left Subclavian a.

Clinical Manifestations

- Small – no symptoms
- Larger L → R: CHF, slow growth, repeated Lower Resp tract infections
- Continuous "machinery murmur" (*other causes of continuous murmur- AV fistula, ruptured sinus of valsalva,aorticopulmonary septal defect, bronchial collaterals, venous hum*)
- peripheral signs of wide pulse pressure may be seen

Treatment

- **Indomethacin, ibuprofen closes PDA by decreasing PGE_1 levels in preterm babies**
- **In term babies, may close by 1 month, if not surgery is required**

Cyanotic Heart Disease

Tetralogy of Fallot

Most common cause of Cyanosis presenting during 3rd week of life

4 components:

- VSD,
- Right ventricular outflow obstruction,
- Right ventricular hypertrophy, and
- "overriding" large aorta

Clinical Manifestations

- Cyanosis due to right to left shunting across VSD and decreased Pulmonary flow
- dyspnea,
- polycythemia,
- squatting spells
- CHF does not occur except when there is associated **anaemia, endocarditis, hypertension**
- "cyanotic spells": periodic cyanosis and agitation – caused by increase in RV outflow tract resistance, increasing the R → L shunt. Last minutes to hours, may resolve or progress to death

Examination:

- clubbing,
- **RV heave felt,**
- **S2 is single,**
- **Systolic ejection murmur** in L upper sternum due to RV outflow obstruction

X-Ray : **boot shaped heart or cor-en-sabot appearance**

ECG – shows RA dilation, RV hypertrophy

Complications: worsening of stenosis with age, infective endocarditis, hemiplegia, brain abscess, severe bronchopneumonia

Treatment

"Cyanotic spells"– Rx by decreasing R → L shunting, by increasing systemic vascular resistance, and dec pulm vascular resistance

Supplemental O2, morphine sulfate,knee-chest position, sodium bicarbonate, propranolol(also for prophylaxis), methoxamine.

Surgery is required in most of the patients.

Palliative surgeries:

- Blalock taussig shunt- pulmonary artery and subclavian artery
- Pott's shunt- PA with descending aorta
- Watersten's shunt- PA with ascending aorta

Truncus Arteriosus

Single arterial vessel, therefore complete mixing of systemic and pulmonic blood usually has ventricular septal defect

Clinical Manifestations

Moderate Cyanosis, CHF develops in weeks

Treatment: Anticongestion = digoxin & diuretics

Surgical repair – close VSD, and make conduit b/n both aorta and pulm artery

d-Transposition of Great Arteries

Aorta arises from RV, Pulmonary A. arises from **LV** – operates in Parallel. Aorta is anterior and to right of pulmonary artery (normally it is posterior and to right)

l-TGA is a corrected TGA (aorta is to the left of PA)

most common cyanotic HD presenting in **neonates who** may have intact ventricular septum

ASD, VSD, or PDA that allows mixing is required for survival

Clinical Manifestions

Cyanosis present from birth and CHF

Right ventricular hypertrophy seen, right axis deviation on ECG, **Egg-shaped heart** on X-ray

Treatment: PGE_1 to keep PDA open in those with intact septum and then emergency Rashkind balloon atrial septostomy

Surgical repair – arterial switch(Jatene's) or Atrial switch(Mustard)

Total Anomalous Pulmonary Venous Connection(TAPVC)

Rare lesion, where all pulmonary venous return is directed to the Right Atrium

May be Supracardiac (most common), Cardiac, Infracardiac or Mixed

Clinical Manifestations: cyanosis and CHF

CXR: figure of eight or snowman appearance

ECG shows right axis deviation and Right Ventricular Hypertrophy

Treatment Rx of CHF

Surgically redirect vessels into Left Atrium Necessary in 1st month of Life

Tricuspid Atresia

No connection b/n RA and RV, leads to hypoplasia or absence of Right ventricle, 90% have VSD (this allows blood to flow from the left ventricle to both the Pulm A. & Aorta

Most also have pulmonic stenosis

The only way for systemic blood to get to the Left side (and therefore the Pulm A. and Aorta is through a Patent Foramen Ovale or ASD

Clinical Manifestations Cyanosis is severe, poor feeding, tachypnea over first 2 weeks of life

ECG – **superior axis and Left Ventricular Hypertrophy**

Treatment PGE_1 to keep PDA open to maintain Pulmonary flow and balloon atrial septostomy, if atrial defect not sufficient Blalock-Taussig Shunt

Ebstein's Anomaly

Extremely rare, where leaflets are displaced into RV cavity. Results in hypoplasia of RV, tricuspid regurg and/or stenosis. 80% have Patent Foramen Ovale, with R → L shunt

Massively dilated RA

Clinical Manifestations

Cyanosis and CHF in first few days of life

heart blocks common

CXR – MASSIVE cardiomegaly, with enlarged RA

Treatment: PGE_1 to keep PDA open to maintain Pulmonary flow, CHF therapy with Digoxin and diuretics

Surgical repair of Tricuspid valve

Hypoplastic Left Heart Syndrome (HLHS)

Most common cause of death from congenital HD during 1st month

Syndrome with: Hypoplasia of LV and Aorta leading to reduced flow through the Left side

Causes L → R shunt at Atrial level & R → L shunt at Ductus Arteriosus

Clinical Manifestations: As ductus close → CHF, moderate cyanosis, tachypnea, pulmonary rales and hepatomegaly

Poor or absent peripheral pulses

Treatment: PGE_1 to keep PDA open to maintain flow to the Aorta from right side and palliative surgery

Coarctation of the Aorta

Constriction of aorta, usually descending at insertion site of ductus.

Clinical Manifestations

Weak femoral pulses relative to upper

Systolic ejection murmur at Apex collateral formation around scapula and lower border of ribs leading to notching of ribs

systemic hypertension

intermittent claudication, pain and weakness

Brachiofemoral delay

Treatment: Balloon angioplasty or surgical end-to-end anastomosis

Aortic stenosis

- left ventricular hypertrophy
- history of angina on effort
- supravalvular aortic stenosis (Williams syndrome) is associated with Elfin facies, mental retardation, dental abnormalities, **idiopathic hypercalcaemia**

Eisenmenger syndrome: occurs in patients with increased pulmonary bloodflow. Over time pulmonary pressures increase as a result or damage to pulmonary vessels.

Increased pulmonary bloodflow-----> pulmonary hypertension------> irreversible pulmonary vessel changes-------> right ventricular hypertrophy------> right to left shunt

The heart disease is inoperable once Eisenmenger syndrome occurs

Congestive heart failure:

Causes: Infants-congenital heart disease, myocarditis, arrhythmias, hypocalcaemia

Older children-rheumatic fever, rheumatic heart disease, myocardial disease, hypertension

Treatment: Frusemide, ACE inhibitors, Digoxin

Acute rheumatic fever:

- seen in the age group 5 to 15 years
- caused by group A beta hemolytic streptococci
- diagnosis by Jones criteria: two major or one major with two minor in the presence of essential criteria
- major criteria: Carditis, migratory polyarthritis, subcutaneous nodules, Chorea, erythema marginatum
- minor criteria: fever, arthralgia, raised acute phase reactants, increased PR interval
- essential criteria: increased ASLO, positive throat culture, recent scarlet fever
- treatment: suppressive agents aspirin or steroids for total of 12 weeks. Steroids preferred if carditis present (full dose for three weeks followed by tapering)
- three weekly injections of Benzathine penicillin lifelong for prophylaxis

Hypertension:

arterial blood pressure above 95th percentile with reference to age, sex and height. Most common cause is secondary to renal parenchymal diseases.

NEPHROLOGY

Development

- Kidney formation starts in fifth week of gestation; maximum nephrogenesis occurs between 20 to 36 weeks and whole number of nephrons formed by 36 weeks
- glomerular filtration starts at 9 to 12 weeks: urine formation
- concentration of urine maximum 700 to 800 mosm/kg at birth; older child, 1200 to 1400 mosm/kg
- renal function matures by two years of age

Common clinical symptoms of renal disease include oedema, hematuria, oliguria, and dysuria.

Edema is characterised by being prominent in morning and periorbital puffiness and later generalised anasarca.

Hematuria is cola colored in glomerulonephritis and bright red in non glomerular causes. Most common cause in children is post-infectious glomerulonephritis.

Oliguria: urine output < 1 ml/kg/hr

Investigations

- Urine routine microscopy: Hematuria is more than five RBCs in centrifuged Sample. More than 10 leucocytes/ HPF abnormal. Proteinuria- < 100 $mg/m^2/hr$- normal; 100-1000 $mg/m^2/hr$- mild to moderate; >1000 $mg/m^2/hr$- heavy or nephrotic range.
- Urine culture- early morning mid stream sample best. Colony count > 100000 taken significant. >10000 significant in symptomatic patients. Any growth significant in suprapubic sample.
- Blood tests: blood urea, 20 to 35; creatinine, 0.4 to 0.8
- Radiology: x-ray, ultrasound, IVP
- Radionuclide scans: technetium labelled DMSA, DTPA, MAG-3. DMSA- structure; DTPA- function; MAG-3- both

Acute glomerulonephritis

Characterised by abrupt onset hematuria, oliguria, oedema and hypertension

Postinfectious:

- Most common.
- Disease of school age children 5-15 years. boys more commonly affected than girls
- Occurs following Group A beta hemolytic streptococcal infection preceding by 1 to 4 weeks; nephritogenic strains-4, 12, 49
- Clinical features include oliguria, oedema, hypertension, cola coloured urine. There is mild proteinuria, blood urea and creatinine may be elevated.
- Serological tests for streptococcal infection are positive in most patients with pharyngitis. ASO and anti-DNAase B are used for diagnosis. Serum level of C3 is characteristically low in 90% patients.
- Management is conservative. Antihypertensives may be required. Diuretic are given for oedema if required. Severe cases may require dialysis.

- Prognosis is excellent. Resolution starts in first week and within two weeks most of the symptoms disappear. Hypertension may take longer to subside.

Rapidly progressive glomerulonephritis

- there is rapid loss of renal function
- histological feature is presence of crescents in more than 50% glomeruli
- chances of chromic glomerulonephritis are high
- Treatment is early intravenous steroids followed by oral prednisolone and cyclophosphamide.

Henoch Schonlein Purpura

- Common vasculitis in children characterised by palpable purpuric rash, arthralgia, abdominal pain and renal involvement.
- Rash is usually on extensor aspects of lower extremities and buttocks
- Renal involvement is usually mild with microscopic hematuria and mild proteinuria; rarely nephritic or nephrotic syndrome can occur. Most patients recover spontaneously, rarely chromic damage occurs.
- Management is supportive. Prednisolone may be tried in some cases

Haemolytic uremic syndrome

- common cause of acute renal failure in children less than 2 to 3 years
- characterised by hemolysis, thrombocytopenia, acute renal failure
- D+ group: preceded by diarrhoea mainly caused by E. coli 0157: H7; I, SHIGELLA DYSENTERY TYPE I (most common in India); D- group: not preceded by diarrhoea
- Following an episode of acute diarrhoea or dysentery, patients show sudden pallor and oliguria. Blood pressure may be high
- Peripheral smear will show features of haemolysis, high reticulocyte counts, and thrombocytopenia. Blood urea and creatinine are elevated
- Treatment is management of renal failure and correction of anaemia. Peritoneal dialysis may be necessary. Residual renal damage can occur in some cases

IGA nephropathy

- commonly causes recurrent episodes of gross hematuria lasting for 2 to 5 days
- preceded by URI
- between episodes mild proteinuria or microscopic hematuria may persist
- Rarely nephrotic or nephrotic syndrome can occur. Some patients may have chronic renal damage

Nephrotic syndrome:

- severe proteinuria (>1g/m2/day), hypoalbuminemia (<2.5 g/dl), oedema, hyperlipidemia
- hematuria, hypertension may be present
- Cause: greater than 90%, idiopathic; Other causes-amyloidosis, SLE, vasculitis, hepatitis B

- based on histology there are two main groups – minimal change nephrotic syndrome and nephrotic syndrome with significant lesions
- *Minimal change disease:* – most common cause in children. Onset generally 2 to 6 years. Clinical features include generalised oedema and oliguria. Hematuria and hypertension are rare. Laboratory features of nephrotic syndrome are present, blood urea and creatinine is usually normal and C3 levels are normal. Response to steroids is characteristically good. Long term prognosis is good with little chances of chronic renal damage. The course is characterised by frequent relapses that generally stop by second decade. Treatment is steroids. Prednisolone is commonly used and the response is good. Diuretics are used if required. ACE inhibitors are preferred as antihypertensives as they also decrease proteinuria. Patients not responding to prednisolone or frequent relapsers may require levamisole, cyclophosphamide or cyclosporine. Common complications include infection: Pneumococcus, gram negative, varicella; renal vein thrombosis; acute renal failure; steroid toxicity.
- *Non-minimal change nephrotic syndrome or nephrotic syndrome with significant lesions*: includes mesangioproliferative glomerulonephritis, focal segmental glomerulosclerosis, and membranous glomerulonephritis. This constitutes 10 to 15% of children with nephrotic syndrome. Hypertension and hematuria commonly occur and response to prednisolone is generally poor. Long-term prognosis is not very good and many patients progress to chronic glomerulonephritis.

Urinary tract infection

Common in children and in smaller children, is frequently associated with vesicoureteric reflux (VUR). In infancy incidence in boys is same as girls. Later: girls> boys. Causes: most common E. Coli. Others: Klebsiella, Proteus, Pseudomonas, Enterococcus. Predisposing factors— vesicoureteric reflux, obstructive uropathy, neurogenic bladder. Clinical features include fever, frequent micturition and dysuria.

Urine microscopy shows > 10 WBC/ cu mm. Urine culture is diagnostic. $>10^5$ organisms per ml is positive in a mid stream clean catch sample; $>10^4$ with symptoms; suprapubic, any growth. Treatment is with appropriate antibiotics and after treating the episode, evaluation of urinary tract needs to be done. Ultrasound is done in all cases. MCU and renal scan are recommended in children less than two years of age.

Reflux nephropathy and Vesicoureteric reflux (VUR)

- retrograde flow of urine from the bladder to upper urinary tract occurs leading to renal damage
- VUR is present in 30 to 35% of children with febrile UTI
- Diagnosis is by MCU which can be done with radio contrast or radionuclide agents
- VUR is graded from I to V (I-reflux into ureters, II-reflux up to renal pelvis, III-dilatation of ureters, IV-dilatation of renal pelvis also, V-massive dilatation of ureters and pelvis)

- Antibiotic prophylaxis for prevention of repeated UTI's is given if VUR is present and in most cases VUR resolves with age.
- Indication for surgery-recurrent breakthrough urinary tract infections, poor compliance/intolerance, appearance of new scars/ persistence of grade 4 and 5 VUR after two years of age. Surgical procedure done is ureteric re-implantation.

Renal tubular acidosis

- There is defect in acidification of urine with resultant hyperchloremic metabolic acidosis, with a high urine pH. Kidney function tests are normal.
- Type I: distal: defective secretion of H+ in distal tubule. Clinical features include failure to thrive, polyuria, polydipsia, hypokalemia and rickets. Investigations show systemic acidosis, hypokalemia and Urine pH>6. Treatment is oral sodium bicarbonate.
- Type II (proximal): it is due to reduced proximal tubular reabsorption of bicarbonate. Acidosis is less severe as the distal mechanisms are intact. It is usually a part of global tubular dysfunction called Fanconi's syndrome when accompanying proteinuria, glucosuria, phosphaturia, uricosuria are also present.

Idiopathic hypercalciuria

Common in children. Manifested as microscopic or gross haematuria and renal calculi. Urine calcium to creatinine ratio greater than 0.2 is diagnostic. Serum calcium is usually normal. Treatment includes salt restriction and thiazide diuretics.

CHILDHOOD MALIGNANCIES

Most common: leukaemia (30%)

Second most common: brain tumours (20%)

Third most common: lymphoma (12%)

leukaemias

- **clonal** proliferation of abnormal hematopoeitic cells.
- Acute lymphoblastic leukaemia is most common
- risk factors: syndromes like Downs syndrome, Klinefelters syndrome, blooms syndrome, ataxia telengiectasia, fanconi syndrome; exposure to ionising radiation; chemotherapy.

Acute lymphoblastic leukaemia

- usual age: 3 to 7 years
- L1 type is most common
- Poor prognostic indicators
  a. age of onset <1 yr or >10 yrs
  b. high initial total leucocyte count (>50,000)
  c. hypodiploidy
  d. 9,22 and 4,11 translocation
  e. response to steroid treatment

clinical features: rapidly increasing pallor, fever, bleeding manifestations. Examination usually reveals lymphadenopathy and hepatosplenomegaly. Peripheral smear will show the abnormal cells and bone marrow examination is confirmatory.

Treatment: response to chemo therapy is generally good. Chemotherapy is given in three phases-induction phase, consolidation phase and maintenance phase. Usual drugs used include vincristine, prednisolone, methotrexate, Cyclophosphamide and L-Asparaginase. Most common cause of treatment failure is relapse. Common sites of relapse are bone marrow, central nervous system and testis.

ACUTE MYELOBLASTIC LEUKEMIA

- prognosis is not as good ALL.
- Morphologically divided into eight types: M0 to M7
- congenital leukemia IS GENERALLY M7 AML
- disseminated intravascular coagulation is a common feature of M3 AML
- gum hyperplasia may be seen in M4 and M5 subsets
- chloromas are localised collections of malignant cells seen in bones like orbit, central nervous system, skin and other sites
- myelodysplastic syndrome is a prolonged pre-leukaemic phases of AML.

NEUROBLASTOMA

- malignant tumour of autonomic nervous system derived from neural crest

- most common intraabdominal solid tumour in children
- most common tumour in infancy
- most common tumour with skeletal metastases
- mostly seen in children less than two years and almost all cases occur by five years
- Common sites are adrenal gland, paravertebral, retro peritoneal, posterior mediastinal and pelvis
- the malignant mass may produce catecholamines leading to systemic features like fever, anxiety, sweating, diarrhoea, hypertension. urinary catecholamines may be elevated .

WILM'S TUMOUR

- most common malignant tumour of kidney
- mostly in children less than five years
- most common presentation is asymptomatic abdominal mass. Hematuria, fever hypertension, abdominal pain may be present.
- Associations: hemihypertrophy, aniridia, genitourinary anomalies, beckewith wiedeman syndrome.

BRAIN TUMOURS

- second most common tumour in children
- around two thirds are infratentorial
- most common are medulloblastomas followed by cerebellar astrocytomas.

RETINOBLASTOMA

- most common primary ocular tumour
- mostly in children less than five years
- bilateral disease is common
- autosomal dominant inheritance, 10% may have family history of retinoblastoma
- earliest sign is Leukocoria

HISTIOCYTOSIS X (langerhans cell histiocytosis)

- infiltration of tissues by abnormal histiocytes , lymphocytes,eosinophils and normal histiocytes.
- birbeck granules are characteristic
- course is unpredictable varying from rapid progression to repeated recurrences to spontaneous resolution
- most common site is skeletal commonly skull bones and long bones. X-ray will show lytic lesions, there may be pathological fractures, vertebral collapse
- other features include seborrheic skin rash, lymphadenopathy, hepatosplenomegaly, chronic ear discharge, lung infiltrates, exophthalmos, gingivitis

Nutrition

Energy requirement: 100 kcal/Kg for the first 10 kgs. Between 10 to 20 kg requirement is 1000 kcal +50 kcal per kg added for the weight above 10 kg. For weight more than 20 kgs, 20 kcal/kg is added to 1500 kcal to estimate the requirement.

Protein requirement:

less than 6 months – 2 g/kg/day
6 to 12 months 1.6 g/kg/day
1 to 3 years 20 g per day,
4 to 6 years 30 g per day,
7 to 9 years 40 g per day and
Thereafter 50 to 70 g per day.

Exclusive breastfeeding should be done till six months of age. After six months complementary feeding should be started and breastfeeding continued and encouraged up to 2 years.

Malnutrition/undernutrition

Indicators of malnutrition

Indicator	*Interpretation*	
Underweight	low weight for age	both acute and chronic malnutrition
Stunting	low height for age	chronic malnutrition
Wasting	low weight for height	acute malnutrition.

WHO classification

The use of Z score or standard deviation score is recommended.

Standard deviation score (SDS)= (observed value-median reference value)/standard deviation. A score of -2 to -3 indicates moderate malnutrition and a score of +2 to +3 indicates overweight. A score of less than -3 indicates severe malnutrition and a score of more than +3 indicates obesity.

WHO classification

	Moderate malnutrition	severe malnutrition
Symmetrical oedema	no	Yes (Edematous malnutrition)
Weight for height	SD score between -2 to -3	SD score < -3 (severe wasting)
Height for age	SD score between -2 to -3	SD score < -3(severe stunting)

IAP classification:

The standard used for reference population was the 50th centile of Harvard standards. This scheme is used in the ICDS programme.

Grade of malnutrition	weight for age of the standard (%)
Normal	> 80
Grade I	71- 80 (mild malnutrition)
Grade II	61-70 (moderate malnutrition)
Grade III	51-60 (severe malnutrition)
Grade IV	<50 (very severe malnutrition)

Mid arm circumference. It is an age independent index of malnutrition. Midarm circumference remains stable between 1 to 5 years at a value of 16 to 17 cm

Less than 13.5 indicates malnutrition

Less than 11.5 indicates severe malnutrition

	Marasmus	**Kwashiorkor**
occurance	More common	Less common, more severe
Edema	negative	Positive
CNS	active, a good appetite	poor appetite with mental apathy
Hepatomegaly	negative	May be present
Prognosis	Better, early recovery	poor(in view of prolonged recovery and more infections)
Appearance	monkey facies baggy pants appearance,	sugar baby appearance skin changes (pigmentation, flaky paint dermatosis) mainly on buttocks, perineum and upper thigh flag sign in hair and hair is easily pluckable

Treatment of malnutrition

- manage complications: hypoglycaemia, hypothermia, dehydration, electrolyte imbalance and infections
- nutritional rehabilitation: Mild to moderate malnutrition- 150 kcal per kg per day and Proteins 3 g/kg/day.

Severe malnutrition- 200 kcal/kg/day and protein: 4 to 6 g/kg/day

Diarrhoea

Most common causes in children are rotavirus and E. coli.

Dehydration status is assessed and classified as no dehydration, some dehydration or severe dehydration according to WHO criteria.

	No dehydration	**some dehydration**	**severe dehydration**
Condition	well alert	restless irritable	lethargic or unconscious
Eyes	normal	Sunken	very sunken
Tears	present	Absent	Absent
Mucosa	Moist	Dry	very dry
Thirst	drinks normally not thirsty	thirsty drinks eagerly	drink poorly, not able to drink
Skin pinch	Goes back quickly	Goes back slowly	goes back very slowly

Oral rehydration therapy (ORT):

Oral rehydration therapy includes ORS, sugar salt solution, lemon water, coconut water, soup, rice water, dal water. Glucose water without salt and fluids like tea and coffee are not ORT.

Composition of ORS

	Low osmolality ORS (new)	Standard WHO ORS (old)
Sodium	75	90
Potassium	20	20
Chloride	65	80
Citrate	10	10
Glucose	75	111
Osmolarity	245	

Patients with no dehydration: replace stool losses with oral rehydration therapy

Patients with some dehydration: 75 ml/kg ORS in the first four hours

Patients with severe dehydration: 100 ml/kg of IV fluid preferably ringers lactate given over 3 to 6 hours.

ZINC is to be given to all children with diarrhoea in the following doses:

Infants < 6 months: 10 mg/day; rest 20 mg/day for 14 days

Down's syndrome

- Most common chromosomal disorder occurring in one in 800 to 1000 babies.
- Caused by trisomy 21 in 94%, translocation in 5% and mosaic in 1%.
- High maternal age has been found to be an important risk factor
- the risk of recurrence is more with translocation especially when they are inherited from the mother
- Features include mental and physical retardation, flat facies, mongoloid slant, epicanthal folds, small nose and flat nasal bridge with low set ears. Hypotonia is seen. These children have a small brachycephalic skull with a flat occiput. They may have clinodactyly, Simian crease, sandal gap.
- They can have cardiac defects most commonly Endocardial Cushion defects. Hirschsprung's disease is also seen. Hearing loss is common. Hypothyroidism is common. They are more prone to develop leukaemia.

Turner's syndrome

45 X0.

- Phenotypically females
- at birth oedema of dorsum of the hands and feet may be present with a short neck
- Later characterised by short stature, delayed puberty, shield like chest with widely spaced nipples, cubitus valgus, and hypothyroidism.
- Left-sided cardiac lesions (AS, COA, MVP), horseshoe kidney, hearing defects
- treatment includes growth hormone therapy and ovarian hormone replacement

Klinefelter's syndrome

(47 XXY)

- Phenotypically males.
- Hypogonadism with small testes and no secondary sexual characters- delayed puberty. These boys are tall and underweight with elongated legs. Gynecomastia or cryptorchidism may be present. They may have mental retardation.